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Letters to the Editor

Visual function impairment in migraine: cerebral versus retinal deficit

To the Editor

Drummond and Anderson (1) detected blurred central vision and depression of visual sensitivity in the peripheral fields in most subjects the day after an attack of migraine with aura. The proposed relationship of such visual field deficit to the residual effect of the migraine aura, i.e., cerebral deficit (1), is weakened by both the absence of neuroanatomically localizing lesions (except one subject with quadrantic homonymous lesion) and the absence of blurring of central vision in 4 of the 10 migraine with aura subjects. The absence of visual field deficit in migraine without aura subjects and relegation of a qualitatively similar increase in visual perception threshold in both varieties of migraine (1) cannot be reconciled with the results of a previous study (2) in which: (i) non-localizing visual field loss was found in approximately one-third of migraine subjects without any visual symptomatology or any indication of ocular involvement, i.e. a cohort of migraine without aura subjects; (ii) 39 of 60 subjects did not demonstrate any form of visual field abnormality, indicating that a sample size of 20 migraine without aura subjects (1) is too small to infer conclusively in this matter; (iii) a greater prevalence of visual field loss with increasing age and duration of disease and the demonstration of replicable lesions reflects a cumulative permanent damage rather than a reversible "functional" deficit. The generalization that peripheral depression of visual sensitivity is consistent with cerebral deficit (1) excludes consideration of another possible pathogenetic mechanism, viz., retinal damage due to low-tension glaucoma (3). Diffuse depression of retinal sensitivity as well as mild reduction in the central vision may precede characteristic visual field changes due to loss of retinal nerve fibre bundles in glaucoma (4). Furthermore, Hupp et al. (5) distinguish between disturbances in peripheral and central vision while elaborating that retinal visual disturbances tend to produce alterations of central vision. Finally, the implication of association of ictally-proximate abnormalities in visual evoked potentials with the migraine aura (1) must be viewed with circumspection because similar aberrations have been demonstrated preictally in a cohort of migraine without aura subjects (6). The flash visual evoked potential reflects transmission of light from the entire retina including fast-conducting axons from the retinal periphery (7); in addition to the proposed greater alteration of non-geniculate pathways, the possibility that some of the aberrations of the visual evoked potential are at the retinal level needs to be resolved (8). Non-localizing visual function impairment, including field deficits (reversible or replicable), cannot be used to emphasize the distinction between migraine with aura and migraine without aura.

Another factor besides migraine-related ischaemia has been suggested to underlie the development of bilateral glaucomatous visual function impairment (2) in patients with unilateral migraine attacks (9); the same considerations apply to the finding of nonlocalizing bilateral reduction in visual field area in migraine patients with unilateral aura (1). The nexus between glaucomatous visual dysfunction and autonomic nervous system dysfunction is unknown; interictal autonomic nervous system hypofunction may allow intermittent (interictal and preictal) transient elevations of the intraocular pressure (in response to a variety of stressful stimuli and situations) that contribute to non-localizing visual function impairment in migraine (9, 10). Absence of significant changes in the intraocular pressure during both the scotoma and pain phases of migraine (11) does not eliminate the possibility of occurrence of interictal retinal/optic nerve head barotrauma (9). Casual tonometry and even more methodical hourly pressure sampling can miss recording periodically elevated intraocular pressure, whereas the mean intraocular pressure and the typical glaucomatous optic atrophy are irrelevant to this situation (12). The applicability of home tonometry (13) is limited in the context of stress-related intraocular pressure elevation (cf. circadian variation) in migraine because (i) stressful events being generally unpredictable, subjects are largely unaware of the threat of impending cephalic disturbance (9), (ii) the onset of stress is likely to trigger neuroendocrine activation including sympathetic hyperfunction, which in turn would tend to maintain a normal intraocular pressure (9), (iii) it is not pragmatic to expect subjects to abruptly cease known usually headache-provoking activities in order to make their own measurements, and (iv) the variable prodrome would render most measurements unrepresentative. Notwithstanding the formidable difficulty in establishing biostatistically significant variation of intraocular pressure in migraine, retinal barotrauma must be considered during elucidation of disease mechanisms for non-localizing glaucomatous visual dysfunction.

Oral administration of propranol, atenolol, metoprolol, and nadolol (4), ventriculo-cisternal perfusion of clonidine (4), and topical application of propra-

nolol (4) and verapamil (14) lower the intraocular pressure. The susceptibility of migraine subjects to develop non-localizing glaucomatous visual dysfunction may be determined by certain risk factors including heredity, myopia, race, sex, diabetes, vascular insufficiency and anaemia (9). Conversely, the ocular hypotensive effects of drugs used for prophylaxis may contribute to the absence of visual field deficit in approximately two-thirds of migraine without aura subjects (2). "Angiospastic" circulatory disturbances of ocular migraine (5) are unrelated to the pathogenesis of the visual function impairment under discussion. Nevertheless, the concept of "occult" intraocular pressure disturbance could serve to rationalize the caveat regarding the use of propranolol in the treatment of anterior visual pathway migraine (15) in the context of widespread use of the drug for prophylaxis; the tendency of propranolol to promote focal vasoconstriction and alter retinal and optic nerve microcirculation may be offset sufficiently by an increase in posterior ciliary circulation consequent to the maintenance of a relatively lower intraocular pressure with elimination of transient elevations.

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Vinod Kumar Gupta, Dubai Police Medical Services, PO Box 12005, Jumeirah, Dubai, United Arab Emirates

To the Editor in reply to Gupta

We mapped the visual fields of migraine with aura and migraine without aura subjects 1 day after an attack, and again 7–10 days later (1). The most striking finding was depression of visual sensitivity in the periphery of the visual fields the day after an attack of migraine with aura. This disturbance had resolved 7–10 days later, and was not observed in migraine without aura subjects. The tests used in our study could not identify the source of the visual disturbance, but we noted that depression of the visual sensitivity in the periphery of the visual fields is consistent with cerebral deficit.

The peripheral visual field deficit the day after an attack of migraine with aura is unlikely to be caused by low tension glaucoma (LTG) for the following reasons: (i) One typical visual field deficit in LTG is a monocular scotoma close to fixation (nerve fibre bundle defect) (2); (ii) other signs of LTG, such as cupping and pallor of the optic disc and mild glaucomatous anterior segment disease, have not been reported in migraine; (iii) the prevalence of migraine in the general population (about 20%) is far greater than the prevalence of LTG (less than 1%) (2). Phelps and Corbett (3) reported that patients with LTG had headaches with or without features of migraine more frequently than other patients with ocular disease or normal subjects. They suggested that optic nerve damage in some patients with LTG might be caused by migraine-related ischaemia. Central scotomas developed more frequently in migraine with aura subjects than in migraine without aura subjects (1, 4); Lewis et al. (5) detected visual field loss in 35% of migraine patients with no other ocular problem (including normal intraocular pressure and optic discs), but did not explore the relationship between the migraine aura and nature of the visual field deficit. In particular, Lewis et al. (5) did not describe the proportion of patients in their series whose headaches were associated with a migraine aura.

Persistent central visual deficit is probably caused by retinal or optic nerve ischaemia in some migrainous patients, and by cerebral ischaemia in others. The mechanism responsible for the transient peripheral constriction of visual fields the day after an attack of migraine with aura (1) is unknown, but the associa-